

while xanthine dehydrogenase is composed of M, L, and R subunits. The proof of this theory must await purification of these substances, although finding electrophoretic variants of xanthine dehydrogenase associated with each locus would indicate that all three are structural genes for this enzyme. We are currently analyzing three electrophoretic variants which we have found.

The idea that the $ma-1^+$ maternal substance is a stable template would also explain the effect of lxd on the maternal effect. This possibility is also being tested by analyzing the amount of the $ma-1^+$ complementation factor during the development of maternally affected flies.

Mittler, S. Northern Illinois University. AET and radiation induced crossing-over in male D. melanogaster.

A re-examination of the problem (Mittler and Hampel DIS 38) of whether AET has any effect upon radiation induced crossing-over in male D. melanogaster indicates that AET does afford significant protection to 9-12 day brood after

irradiation. Adult males 2-16 hr. old heterozygous for $ru\ h\ th\ st\ cu\ sr\ e^s\ ca$ were injected with 1×10^{-7} l. of 30 mg. AET/10ml buffered to pH of 7 and irradiated with 2000r and then back crossed to homozygous "rucuca" females at ratio of 1 male to 3 females. The males were transferred to new group of females every 3 days.

Number Males	9-12 day brood			12-15 day brood		
	<u>Crossover</u>	<u>Non</u>	<u>% Crossover</u>	<u>Crossover</u>	<u>Non</u>	<u>% Crossover</u>
120 Treated	57	8,502	.666	.54	11,361	.473
101 Control	49	5,025	.966	29	4,187	.688

Glassman, E. University of North Carolina. Interaction of $ma-1$ and lxd in the synthesis of pyridoxal oxidase and xanthine dehydrogenase.

The maroon-like ($ma-1$) and rosy (ry) eye color mutants lack detectable amounts of xanthine dehydrogenase. A strain which is mutant at a third locus (lxd) has only 20 to 25% normal amounts of this enzyme. Analysis of lxd flies reveals that the level of CRM (the suspected

product of the ry^+ locus) is the same in lxd as in wild type, while pyridoxal oxidase (the suspected product of the $ma-1^+$ locus) is absent in lxd . Complementation tests in vitro are in agreement with these findings; thus there is no $ma-1^+$ complementation factor in extracts of $lxd\ ry$ flies while the ry^+ complementation factor seems to be present in normal amounts in $ma-1; lxd$.

It seems evident that lxd is deficient in those molecules which are also specifically deficient in $ma-1$ (such as pyridoxal oxidase and the $ma-1^+$ complementation factor) while the molecules missing in ry (such as CRM) seem to be present in normal amounts in lxd . Thus, the normal production of the $ma-1^+$ complementation factor and pyridoxal oxidase must be due to the interactions of the lxd^+ and the $ma-1^+$ loci. The paradox of the presence of substantial, though less than normal, amounts of xanthine dehydrogenase in lxd in spite of the absence of the product of the $ma-1^+$ locus can be resolved by various hypotheses. One can postulate that the ry^+ , the $ma-1^+$, and the lxd^+ loci code for three different polypeptide chains, designated R, M, and L, respectively. These polypeptides produce different enzyme activities by polymerizing with each other in different ways. Thus, a polymer of the M and L polypeptides will have pyridoxal oxidase activity, while any polymer containing R polypeptides will have CRM activity. Only a polymer containing R, M, and L polypeptides will have xanthine dehydrogenase activity. On this basis, the in vitro complementation reaction between $ma-1$ and ry extracts would be visualized as an interaction between two large polymers containing ML and R subunits, respectively. The deficiencies observed in lxd flies would be due to a defective L polypeptide which can still react in vivo to yield some xanthine dehydrogenase activity, but which is too defective to yield active ML polymers (pyridoxal oxidase). It is of interest that pyridoxal oxidase has a molecular weight almost as great as that of xanthine dehydrogenase (about 250,000).

Alternatively, one can view the *lxd* gene as a regulator of the activity of either the *ma-1*⁺ gene or its product. In this case, the pyridoxal oxidase would be a polymer composed of only M polypeptides, CRM would again be any polymer containing R polypeptides, while xanthine dehydrogenase activity would be a polymer of M and R polypeptides. Another possibility is that only the *ry*⁺ locus codes for a subunit of xanthine dehydrogenase, while the products of the *ma-1*⁺ and *lxd*⁺ loci are regulators or activators, not only of the R polypeptide which forms xanthine dehydrogenase, but also of other polypeptides which form pyridoxal oxidase and other unknown enzymes.

Muller, H. J. Indiana University. An improved stock, "vix," for scoring visible mutations that arise at specific loci in the X of the female.

The fertile females of this stock have X-chromosomes of the same composition as those of the "Maxy-v" stock (number f30 of the 1957 Bloomington stock list in DIS 31), except that in this stock the recessive genes *ptg* and *oc* and the dominant *B* are in the X-chromosome having

most of the recessive mutants, while the balancing X-chromosome is supplied with *lz*^S instead of *ptg oc*. Moreover, the fertile females contain a Y-chromosome, of normal type. The X-chromosome of the fertile males has the composition *y oc ptg lz f*. As in the stocks "jynd" and "plond" (f72 and f88, respectively, of DIS 31), but even more so because of the additional pressure of the *B*^{M1} inversion in one X, the combination of heterozygous inversions in the presence of a free Y causes a high frequency of non-disjunction of the X's, with resultant matroclinous daughters, bearing both the mother's X's, that can be scored for visible mutations arising in the originally wild-type loci of the non-multiple-recessive X, while at the same time the only fertile sons (in this case, in fact, the only viable sons) are patroclinous. One kind of disjunctionally produced daughter, of phenotype *y oc ptg B f*, is sterilized by *oc*, while the other kind, of phenotype *lz B*⁺, is rendered highly infertile by the *lz/lz*^S combination and, if it should breed, would produce virtually no crossovers of a kind that would disturb the genetic system.

The reproducing individuals are as follows:

males: $Y^+/y \text{ oc ptg lz f}$

females: $\frac{Y^+/1J1 \text{ sc}^{J1(+)} \text{ In49 v lz}^S \text{ B}^{M1}, \text{In}}{y \text{ sc}^{S1} \text{ car odsy B f g}^2 \text{ dy v ras}^2 \text{ ptg oc sn}^3 \text{ ct}^6 \text{ cm rb ec w pn l sc}^8$

Any mutant or suspected mutant daughter arising non-disjunctionally can be bred with her brothers for verification and for establishing a balanced stock. The phenotypes of the disjunctionally arising daughters present combinations of peculiarities that keep them from being mistaken, even at first sight, from mutant daughters of non-disjunctional origin.

The "vix" stock not only has the advantage over "jynd" and "plond" of giving no disjunctional males. Its normal Y gives rise to more non-disjunction of the X's (more than half of the daughters being non-disjunctional) than does the ring-Y (*Y*^{Lc}) of "jynd". Moreover, the *Y*⁺, unlike the *y*^L of "plond", leaves uncovered the deficiency of the left end of the X that arises when, occasionally, the X's undergo acrocentric attachment with one another, or when one of them exchanges with the Y (see the Valencias and Muller in DIS 23:99-103); it thereby results in the death of these exceptions which when viable are difficult to recognize for what they really are. It is also to be noted that the homozygosity for vermilion of the parental and non-disjunctionally produced females, which was recommended by Altenburg, makes the other eye color mutants here in question more conspicuous. That is, it sensitizes the observer, as it were, to these changes, and although the locus of vermilion is thereby sacrificed so far as the finding of mutations to *v* is concerned, more is gained than lost by the greater ease and reliability of the scoring for these other loci.